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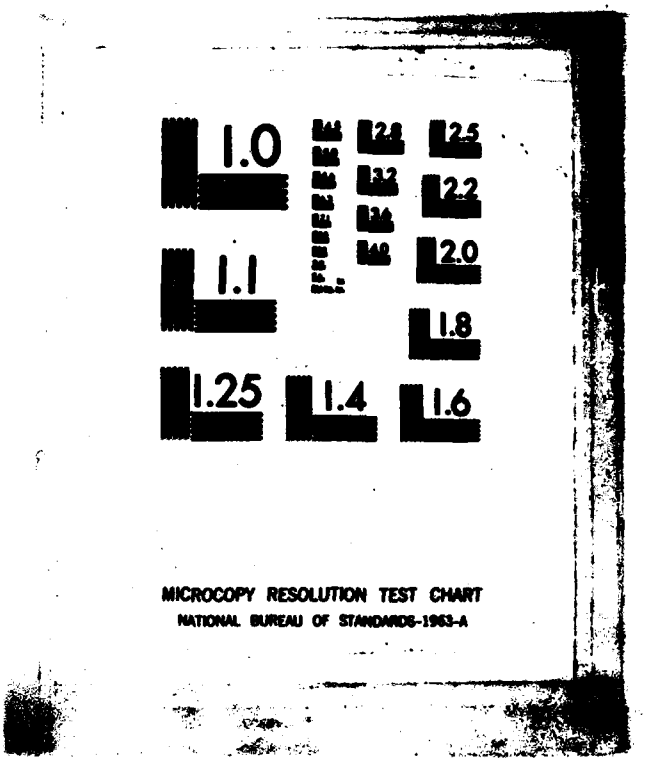
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LABORATORY NOTE: EFFECT ON SLEEP LATENCY OF PRE-SLEEP AEP PROCEDURES

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REPORT NO. 84-17

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LABORATORY NOTE:
EFFECT ON SLEEP LATENCY OF PRE-SLEEP AEP PROCEDURES

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To expedite communication of our research, this is a preprint of a paper submitted to Sleep and should be cited as a personal communication.

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SUMMARY

In a 12-night study of the effects of l-tryptophan in poor sleepers, waking auditory evoked potentials (AEPs) were obtained prior to lights out on the third placebo-baseline night and the fifth treatment night. Sleep latencies were significantly shorter on both AEP nights. The components of the AEP procedure may facilitate sleep onset by promoting relaxation and lowering psychophysiological arousal level in poor sleepers.

INTRODUCTION

Non-pharmacological techniques designed to aid sleep onset in insomniacs are usually intended to reduce psychophysiological arousal levels and/or cognitive activity prior to sleep onset. The sleep-promoting effects of repeated presentations of monotonous auditory stimuli have been reported (1-5). In this laboratory note, I describe the effects on sleep onset time of our laboratory procedure for recording pre-sleep auditory evoked potentials (AEPs) from poor sleepers. These data were obtained during a 12-night sleep laboratory study of the effects of l-tryptophan (3 grams) on sleep, performance, and arousal threshold.

METHODS

Subjects were 20 male laboratory-qualified poor sleepers (age 20.3±2.4 years) who met both subjective and EEG criteria previously described (6). All showed EEG-recorded sleep latencies of 30 minutes or longer on a sleep laboratory screening night (Night 1). Ten subjects received placebo on three placebo-baseline nights (Nights 2-4), l-tryptophan (3 grams) on six treatment nights (Nights 5-10), and placebo on two withdrawal nights (Nights 11-12). A parallel group of 10 subjects received placebo on Nights 2-12. Lights out (LO) was at 2200 and the morning awakening occurred at 0530. Sleep was recorded and scored according to the usual procedures (7). The details of the 12-night protocol are summarized in Figure 1 and described in detail elsewhere (6).

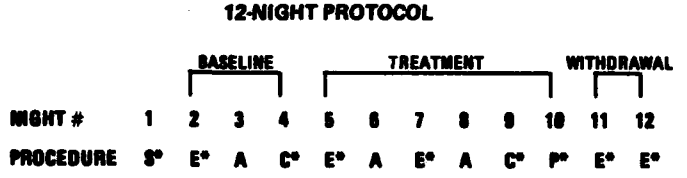

Figure 1

Protocol for the 12-night l-tryptophan study. Procedure code: S=screening night; E=all-night EEG for sleep stage scoring; A=auditory arousal thresholds obtained; C=auditory evoked potentials obtained; P=performance batteries administered during awakenings from sleep; *=morning performance testing.

AEPs were recorded on the third placebo-baseline night (Night 4) and fifth treatment night (Night 9). On these nights, subjects went to bed approximately 10 minutes earlier than on other study nights. They were instructed to relax, lie quietly, and focus their eyes on a black "X" taped onto the ceiling. Clicks (105 dB, 73 dB SPL) were delivered every 15 seconds for 10 minutes through a loudspeaker positioned approximately 46 centimeters above the subject's head. At the end of the collection of awake AEPs, the subject was told "good night" and the room lights were switched off.

RESULTS

Sleep latency data are illustrated in Figure 2.

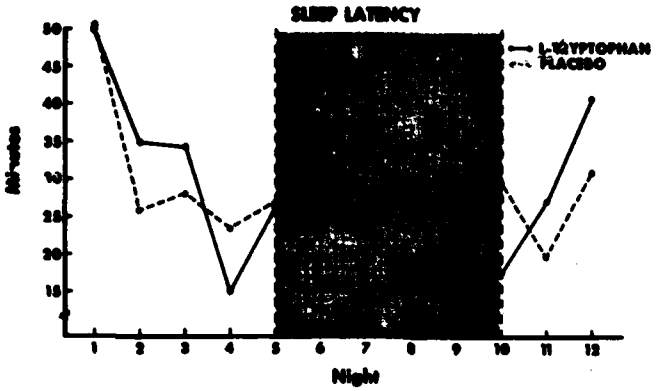

Figure 2

L-tryptophan study: mean sleep latencies for all study nights.

Inspection of mean data for each night indicated that sleep latencies were substantially lower on AEP nights. Over all subjects, sleep latency was significantly reduced on the baseline AEP night compared to the mean sleep latency for the other two baseline nights (19.1±10.4 versus 30.7±15.2 minutes, t_{19}=3.23, p<.004, two-tailed). In the placebo group, sleep latency on the treatment AEP night was significantly lower than the mean latency for the other treatment nights (14.4±8.0 minutes versus 28.1±19.7 minutes, t_9=2.39, p<.05, two-tailed).

DISCUSSION

In this post-hoc analysis of sleep latency data, pre-sleep AEP procedures were found to reduce sleep onset time. This finding is consistent with previous reports of the sleep-facilitating effects of repetitive auditory stimuli (1-5). However, the described procedure differed somewhat from the earlier protocols in that clicks were turned off at LO and did not continue through the awake period from LO to sleep onset. The argument could be made that the AEP effect on sleep latency was due solely to the fact that our subjects went to bed 10 minutes earlier on AEP nights. Since we were not systematically exploring the effects of the AEP procedure, our study design did not include a control for this factor. I note, however, that these laboratory-qualified poor sleepers fit the diagnostic criteria for Disorders of Initiating and Maintaining Sleep, Psychophysiological, Persistent (8). They characteristically report becoming less sleepy rather than more sleepy when lying awake in bed, and they complain of restlessness, ruminative thoughts, and unsuccessful attempts at "trying to fall asleep". Going to bed earlier with the instruction to lie awake should not by itself facilitate sleep onset in psychophysiological insomniacs. Other aspects of the AEP procedure, though, may

result in a psychophysiological relaxation which is of benefit in this form of sleep-onset insomnia. Bohlin (1,2) has suggested that the facilitation of sleep onset by repetitive auditory stimuli was associated with relaxation, as demonstrated by decreases in skin conductance level and frequency of spontaneous skin conductance responses.

The pre-sleep click procedure as described here is probably not a clinically useful technique for the treatment of sleep-onset insomnia. In a previous study (6), the effects were less marked, although, again in that study, the placebo group had its shortest sleep latency on the second AEP night. However, the components of the AEP procedure--relaxing, lying quietly, and processing of a repetitive stimulus--are reminiscent of the counting of sheep and are elements in many behavioral and cognitive techniques taught to improve pre-sleep habits in poor sleepers. It is also important to note that, in sleep laboratory research, any pre-sleep procedures must be carefully evaluated to identify unsuspected effects on sleep latency and other dependent variables.

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In a 12-night study of the effects of l-tryptophan in poor sleepers, waking auditory evoked potentials (AEPs) were obtained prior to lights out on the third placebo-baseline night and the fifth treatment night. Sleep latencies were significantly shorter on both AEP nights. The components of the AEP procedure may facilitate sleep onset by promoting relaxation and lowering psychophysiological arousal level in poor sleepers.

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